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ABSTRACT

This paper examines the theoretical and empirical roles of income poverty and race in the determination of infant mortality differentials in the United States. A basic model of the process of infant mortality is conceptualized. It outlines a theory of the influence of a variety of biological, social, and economic factors upon the risk of infant death. Using national data gathered in 1964-65, estimates are made, for both the black and white populations of the United States, of the relative impact of these factors upon infant death. A key feature of this analysis is the application of methods for the analysis of multidimensional contingency tables. These techniques are firmly grounded in statistical theory, allow for rather sophisticated tests of multivariate hypotheses, and are easily interpretable in terms of the relative risks of death observed in various subpopulations. Within the white population, income poverty is found to be associated with substantial differences in the risk of both neonatal and postneonatal mortality, independent of the birth weight of the infant. Black-white infant mortality differentials are examined, and although socioeconomic differences contribute to these mortality differentials, the high incidence of low birthweight infants among the black population contributes most to this disparity. Improved prenatal care is suggested as one possible mechanism which may serve to eliminate these difference. (Author/AM)

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POVERTY, RACE, AND INFANT MORTALITY IN THE UNITED STATES

Steven L. Gortmaker

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Poverty, Race, and Infant Mortality in the United States

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ABSTRACT

This paper examines the theoretical and empirical roles of income poverty and race in the determination of infant mortality differentials in the United States. A basic model of the process of infant mortality is conceptualized, which outlines a theory of the influence of a variety of biological, social, and economic factors upon the risk of infant death. Using national data gathered in 1964-65, estimates are made for both the black and white populations of the United States of the relative impact of these factors upon infant death. A key feature of this analysis is the application of methods for the analysis of multidimensional contingency tables: these techniques are firmly grounded in statistical theory, allow for rather sophisticated tests of multivariate hypotheses, and are easily interpretable in terms of the relative risks of death observed in various subpopulations.

Within the white population, income poverty is found to be associated with substantial differences in the risk of both neonatal and postneonatal mortality, independent of the birthweight of the infant. Prenatal care--perhaps the major form of health care influencing infant death and disability--has been found to exert its impact mainly via its influence upon subsequent birthweight of the infant. These results strongly suggest some of the limits of health care in reducing economic differentials.

Black-white infant mortality differentials are examined, and although socioeconomic differences contribute to these mortality differentials, the high incidence of low birthweight infants among the black population contributes most to this disparity. Improved prenatal care is suggested as one possible mechanism which may serve to alternate these differences.

Poverty, Race, and Infant Mortality in the United States

1. INTRODUCTION

Differentials in infant mortality rates between population groups have long been a topic of interest to social scientists. In the latter half of the nineteenth century, for example, Marx described in detail the differentials in infant mortality existing across registration districts in England; they ranged from a low of 70 per 1,000 to a high of 250 per 1,000 in the most heavily industrialized areas of the country (Marx 1967, pp. 397-98). Such differentials were surely in large part a result of the terrible conditions under which the exploited working-class population lived. Income poverty, poor sanitation, poor health care, inadequate nutrition, and mothers who were forced to work because of their poverty, and thus neglect their children were all too common circumstances into which children were born.

Although such extremely high absolute levels of infant mortality are no longer found in the industrialized countries of the world, class differentials still exist. In 1964-66 in the United States, for example, it was found that black infants born to parents earning less than $3,000 per year experienced a death rate of 42.5 per 1,000 while white infants born to parents earning $10,000 or more per year experienced a death rate of 19.4 per 1,000 (National Center for Health Statistics 1972). These differentials take on added significance when it is realized that death rates during the first year of life in the United States are exceeded only by death rates to those aged 65 and older (Statistical

Abstract of the United States 1974). Old age and infancy are the times of greatest risk to human life; the magnitude of these observed differentials is thus considerable. However, a consciousness of the meaning of these differentials may not be widespread throughout a population--only one in fifty parents of new infants experience an infant death in a given year. A better idea of the number of lives involved may be estimated by applying the mortality rate experienced by white infants born to parents earning $10,000 or more per year to black infants born to parents earning less than $3,000. If the black infant population could have experienced the white force of mortality, approximately 8,100 more infants would have survived during a given year.

The mere existence of differentials by social class, however, does not constitute a satisfactory explanation of these differences. We know that there are many facts of life related to stratification differentials which may also be related to infant deaths, and vice versa (Shapiro, Schlesinger, and Nesbitt 1968; Kessner et al. 1973; Mechanic, 1968); thus we need to control for confounding factors within the context of a theory if convincing tests of these relationships are to be made.

Methodological Approach

The methodological approach of the research reported here is to construct and estimate multivariate structural equation models of infant death based upon samples of births and infant deaths (defined as occurring during the first year after birth) in 1964-65 in the United States (NCHS 1972). Three aspects of this approach are particularly noteworthy. First, it can be noted that although systematic experimental trials (including randomized assignment to treatment) offer the most desirable evidence for imputing causality to factors of interest, a variety of

problems make them inappropriate for the research reported here. Ethical problems with the manipulation of life chances, as well as cost considerations in the alteration of stratification characteristics of a population combine to make the present observational approach quite attractive. By statistically controlling for a variety of confounding factors simultaneously, the present research should offer some reasonable estimates of effects. It should also be acknowledged that quite a large amount of natural variability exists within the population studied here (the United States), providing the researcher with considerable numbers of situations to study. In particular, it provides the present research with the chance to look at the extremes of the stratification system, for, as will be discussed later, it is under situations such as extreme poverty that we expect effects to appear.

Second, the data used in the research reported here is somewhat unique in that it comes from national random samples of infant births and deaths in 1964-65 in the United States (NCHS 1972). Most previous research looking at a variety of factors influencing infant mortality (including stratification variables) has depended upon more localized samples, such as New York City (Kessner et al. 1973), upper New York State (Chase 1964), Baltimore (Shah and Abbey 1971), and Wisconsin (Slesinger and Travis 1975). No problems of generalizing to the total United States population are encountered with the present data.

Finally, many of the statistical models used in the past to analyze survey data such as that presented here have suffered from a variety of defects. The work reported here utilizes relatively recently developed log-linear models for the analysis of multidimensional

contingency tables, which provide quite satisfactory estimates and tests of hypotheses. The properties of these models are discussed in more detail later. Furthermore, these statistical models are developed upon the basis of a theory of the process operating; in particular, a recursive model of the process of infant mortality is developed and tested. Illustrative diagrams are utilized, whereby variables and their relationships are displayed; these diagrams serve as useful guides to the theory being tested in much the same way that path diagrams have proven helpful in the conceptualization of social phenomena (Duncan 1966). The usefulness of this approach lies in the researcher's ability to concisely display the causal assumptions thought to be operating, and to explicitly test theories based upon these assumptions. Additionally, through the estimation of structural equations, one gains insight into the relative magnitudes of effects operating. This type of information can be valuable for indicating variables worthy of future, more detailed study. One needs only to begin reading the voluminous literature concerning factors implicated in infant mortality to realize that some fundamental causal models certainly need to be specified and tested if useful, cumulative research in the area is to proceed. The present research hopes to offer some first steps in that direction.

The approach used here to estimate the impact of factors operating within a population, of course, is now well established in the social sciences. More important than statistical estimates, however, are the particular theories which define the models to be tested, and which give meaning to the resulting statistics.

2. THE CONCEPTUAL MODEL

A stratification system is characterized by the fact that certain valued resources and ascribed characteristics are differentially distributed across the population of individuals under study; among these characteristics are income, educational attainment, and racial identity. Much of current stratification research (e.g., status attainment research) is concerned with analyzing the attainment process by which individuals become located or locate themselves into the achieved statuses listed above (Blau and Duncan 1967). Other research upon this distributional aspect of stratification theory attempts to explain how distributions of these characteristics arise within different populations. The focus of attention here will be upon assessing some associated consequences of these differentially possessed characteristics for individuals, i.e., is stratification hazardous to one's health? Our concerns will be with this question, particularly: To what extent are poverty and race hazardous to an infant's health?

Studying the consequences of the life situations defined by stratification systems is no easy matter. The rareness of death itself adds considerable difficulty to the task of making precise estimates of how life chances are distributed throughout a population.[1] One fact of infancy makes our task somewhat easier: an infant's own motivations are presumed to have little impact upon its chances for survival--all influences should thus come from its parents and the surrounding environment.

Quite a large variety of environmental and parental influences upon infant death have been researched in the medical, medical-sociological,

and demographic literature. Among stratification factors which have been identified as being related to infant death are parental income, education of mother, education of father (NCHS 1972), occupation of father (Chase 1964), and racial-ethnic identity (Shapiro, Schlesinger, and Nesbitt 1968, p. 24). Very often in the literature, however, little theoretical rationale is given by which such associations with stratification factors may be interpreted. The interpretive theories explicated here will focus upon two components of infant mortality and their relationships with poverty and race: neonatal mortality, or deaths to live-born infants which occur during the first four weeks of life, and post-neonatal mortality, or deaths which occur during the remainder of the first year of life. Neonatal and postneonatal death rates are defined as the number of neonatal or postneonatal deaths per 1,000 live births. It should be noted that, by definition, in any given year, the neonatal and postneonatal mortality rates sum to the infant mortality rate for that year.

One important characteristic of the distinction just made is that different causes of death predominate in the neonatal and postneonatal periods. Neonatal mortality tends to be dominated by death classifications such as immaturity of the infant, postnatal asphyxia and atelectasis, birth injuries, and congenital malformations—in general, causes of death where the "influence of conditions present before the birth or that occur during the birth predominate" (Shapiro, Schlesinger, and Nesbitt 1968, p. 27). Alternatively, we may note that during the "postneonatal period, the infectious diseases, particularly pneumonia and influenza, dominate

as causes of death. Also, accidents begin to assume a major role in mortality" (Shapiro, Schlesinger, and Nesbitt 1968, p. 27).

Both of these components of infant mortality may be differentially related to environmental factors which influence the health of the infant. In particular, deaths to infants with congenital malformations are often unrelated to the environmental conditions under which an infant develops (Gruenwald 1974). Deaths due to the immaturity of the infant have been similarly described as being related in little known ways to the environment of the developing fetus (Morris and Heady 1955; Shapiro, Schlesinger, and Nesbitt 1968, p. 92). Many deaths due to infectious diseases such as pneumonia and influenza, on the other hand, could certainly be prevented through correct medical and home care, and thus should be strongly influenced by environmental factors. These rough distinctions imply that the effects of factors such as poverty are very likely strongest in the postneonatal period.

Studies of the relationships between aspects of socioeconomic status and infant mortality have focused in the past upon this distinction, and have come to two general conclusions. First, a tremendous change has taken place in the last half-century in the distribution of deaths into these two categories. It may be estimated that in 1918 44% of all infant deaths occurred during the neonatal period, and thus 56% occurred during the postneonatal period (National Office of Vital Statistics 1920). In contrast, the 1974 distribution indicates that 77% of all infant deaths occurred during the neonatal period, and only 23% occurred during the postneonatal period. (National Office of Vital

Statistics 1975.) This decline in the relative size of the postneonatal component has been interpreted to mean that the importance of stratification factors in the infant mortality process has declined over the last 60 years; a changing relationship has thus been hypothesized (Morris and Heady 1955; Willie 1959; Stockwell 1962).

Second, studies of relationships between aspects of socioeconomic status and infant mortality have emphasized the significance of income in the determination of relative risks (Anderson 1958; Willie 1959; Willie and Rothney 1962; Stockwell 1962), although the rather small contemporary relationships found have led these authors to emphasize the changing nature of infant mortality, and thus to downplay the current significance of such variables in the determination of infant death. Because these studies used small, local samples, highly aggregated units of analysis (such as census tracts), and rather imprecise measures of the strength of observed relationships, however, this emphasis may seem questionable.

More adequate recent data, such as the National Center for Health Statistics followback surveys (1972), indicate quite significant differentials in infant mortality rates by income and race; unfortunately, no distinction is made in their analysis between neonatal and postneonatal death and few controls for confounding variables of interest are made. Other recent results based upon more local samples, but utilizing more sophisticated techniques of analysis, have demonstrated significant relationships between the socioeconomic status of census tracts and the increased risk of postneonatal mortality, when the effects of other variables are held constant (Shah and Abbey 1971); and, a significant

partial effect of education of mother upon infant mortality has been demonstrated (Slesinger and Travis 1975). No income measures were included in these studies.

Research until now has thus demonstrated the existence of significant differentials in the risk of infant death by a variety of indices of socioeconomic status, within a variety of locales, and statistically controlling for a variety of confounding variables. Yet, a precise explication of the roles of poverty and race in the infant mortality process in the United States remains to be accomplished. The present research aims to correct these past deficiencies.

Some specific theories behind the significance of poverty in the infant mortality process may be briefly summarized: the fact of income poverty may be conceptualized as exercising a constraint upon many areas of parental and infant activity. These constraints may manifest themselves in the form of poor housing, poor sanitary facilities at home, lack of adequate food; inadequate prenatal, hospital or postnatal care; lack of transportation facilities—meaning difficulty in obtaining needed services; and finally, those in poverty may be more vulnerable to the experience of stressful situations. All of these factors presumably can have damaging effects upon the health of the infant.

The kind of health care received by the mother and infant understandably comprises a significant component of these influences. It is thus useful to distinguish conceptually between those effects of poverty, and in fact other aspects of stratification which influence infant death indirectly through health care variables, and those effects of poverty which influence infant mortality independently of these aspects of health

care organization and delivery. From a public policy perspective, health programs for mothers and infants constitute a significant public expenditure, although the amount spent upon children per capita is considerably less than that spent upon the adult population (Haggerty, Roghman, and Pless 1975, p. 37). In addition, any increase in expenditures for improved health care for mothers and infants may be found to be small indeed when contrasted with some of the long term costs of providing care to chronically handicapped children whose condition may be related to poor health care prior to and during the birth process. Most important, however, is the fact that the delivery of health care may be viewed as one intervening factor in the infant mortality process which may be changed, and which thus may be used as a mechanism for reducing death and disability in the future.

One further point needs to be made concerning the impact of income poverty upon infant death. As Mechanic has suggested in his review of literature, which finds little relationship between socioeconomic status and infant death, researchers should perhaps

> . . . . conceptualize the relationship between socioeconomic status and infant mortality in some fashion other than as a linear relationship. Thus, the relationship may hold only when deprived social and cultural groups are involved, so that we might expect a curve characterizing the relationship to reach a plateau at a level of income which might be thought of as relatively low [1968, p. 246].

Such a view is taken in the present research; I specifically hypothesize that it is among those in extreme poverty that a high risk of infant death is most likely to be found.

Racial differences in infant mortality have also been a persistent feature of life in the United States; although both black and white

populations have experienced tremendous reductions in the risk of infant death over the past 50 years, black infant mortality rates still lag significantly behind the corresponding white rates. In 1974, for example, black infants experienced a force of mortality equivalent to that experienced by white infants in 1953 (National Office of Vital Statistics 1975; 1954).

A variety of fundamental differences may be recognized which distinguish black from white society in the United States, and which likely have significant impact upon the production and reproduction of black-white infant mortality differentials. One striking difference may be found in the health care personnel available to blacks. In urban centers such as Chicago, Los Angeles, and Baltimore, less than half as many doctors are available to the urban poor--and typically black--residents as are available to their more affluent neighbors (Norman 1969, pp. 119, 89).

Besides the fact of their segregation from health services, blacks in the United States further experience a variety of social and economic disadvantages relative to whites; among these are differences in income, occupational and educational attainment, as well as the facts of racism and discrimination. The consequences of these differences for observed differentials in infant mortality have not been convincingly analysed. Some efforts in this direction will be made in the present research.

One major finding with respect to black-white differentials in infant mortality concerns the extent to which low birthweight plays a significant role in accounting for observed differences (Shapiro, Schlesinger, and Nesbitt 1968, pp. 53, 84; Susser, Marolla, and Fleiss

1972; Habicht et al. 1974; Slesinger and Travis 1975). These studies all tend to agree that much of the black-white differential in infant mortality may be attributed to different birthweight distributions between the races.

Birthweight, however, must also be considered an outcome measure of the infant mortality process, and few well-substantiated explanations have been offered for these large, and persistent, black-white differences. One explanation forwarded concerns the nutritional status of black mothers (Bergner and Susser 1970; Habicht et al. 1974), although little direct evidence from contemporary experience in the United States supports the pervasive importance of this factor. Some of the earliest studies into nutritional factors associated with low birthweight infants were natural experiments during World War II; a study based in Holland, for example, reported a decline in mean birthweight of 200 gm (Smith 1947), and a mean decline of 500 gm was reported during the siege of Leningrad (Antonov 1947). More recently, Habicht et al. (1974) reported a successful experiment in Guatamala in which mothers were given food supplements and birthweights of their infants subsequently increased. The extent to which these experiences may be translated into the United States, however, is difficult to assess.

One factor which has not been extensively explored in these regards is the quantity and quality of prenatal care received by black mothers, and the corresponding potential that prenatal care may hold as a mechanism for the reduction of the incidence of low birthweight infants. Recent research with New York City data, for example, demonstrates the close connection between mothers who do not obtain adequate prenatal care, and the subsequent low birthweight of their infants (Kessner et al. 1973, p. 38; Gortmaker 1977, Chapter V). It should be noted, however, that

prenatal care often monitors mothers' diets, and thus nutrition may constitute a significant component of this effect.

Among other factors associated with infant death, three stand out as quite significant (Shapiro, Schlesinger, and Nesbitt 1968) and will be included in the present analysis. These are age of mother, birth order of the child, and the previous pregnancy history of the mother. These may be seen as confounding factors which may obscure the true relationship of the postulated stratification factors to infant mortality.

Age of mother has long been associated with differential chances for infant survival, although the specific mechanisms operating are still largely unclear. It has been rather well established that certain birth defects, such as chromosomal abnormalities, do occur more often to older women (Apgar 1970). The age of younger women has likewise been associated with increased risk of infant death, although recent research indicates that the effect of a young woman's age upon infant mortality may in fact be due to the relatively lighter weight of young mothers and the resulting greater chances of having a low birthweight baby (O'Sullivan et al. 1965; Rush, Davis, and Susser 1972). One result of the present research may be a chance to test the efficacy of these latter two explanations: by controlling for birthweight of the infant, we will be able to assess the significance of both direct effects of age as they affect infant death, and indirect effects as they operate through the birthweight of the infant.

Birth order of the infant has also long been associated with differential chances of death, and here theories as to why this occurs are even less common. Generally, increasing risks have been reported for increasing birth orders (Shapiro, Schlesinger, and Nesbitt 1968, pp. 60-61),

although such analyses rarely take into account many variables of interest. One suggested interpretation is that increasing family size leads to increased chance of the spread of infectious disease, as well as increased strain on parental care, thus leading to increased postneonatal mortality (Benjamin 1965, p. 46).

Finally, it should be noted that mothers with a previous history of pregnancy loss or who experienced a previous infant death have been found to exhibit higher than average rates of infant death in their current pregnancy (NCHS, 1973). A possible explanation here is the hypothesis that "certain women may be reproductively more efficient, because an undue number of pregnancy losses appear to be concentrated in a relatively small group of women, and the type of loss tends to be repeated" (Kessner et al. 1973, pp. 118-19). We will thus control for this variable in the analyses that follow in order to rule out the hypothesis that observed relationships may be due to such a biological mechanism.

Other aspects of stratification, such as educational attainment, occupy a less well-defined theoretical position in the infant mortality process. The present research will include both educational attainment of mother and father in subsequent analyses in order to control for the effects of a variety of nonfinancial characteristics of the family environment, such as knowledge of health practices, or the existence of a more general cultural isolation.

All of the variables discussed thus far as being conceptually important in the infant mortality process can be represented diagrammatically as in Figure 1. We thus assume the stratification position of the parents to be causally prior to events to their right in the diagram, and the concepts located between these stratification variables and

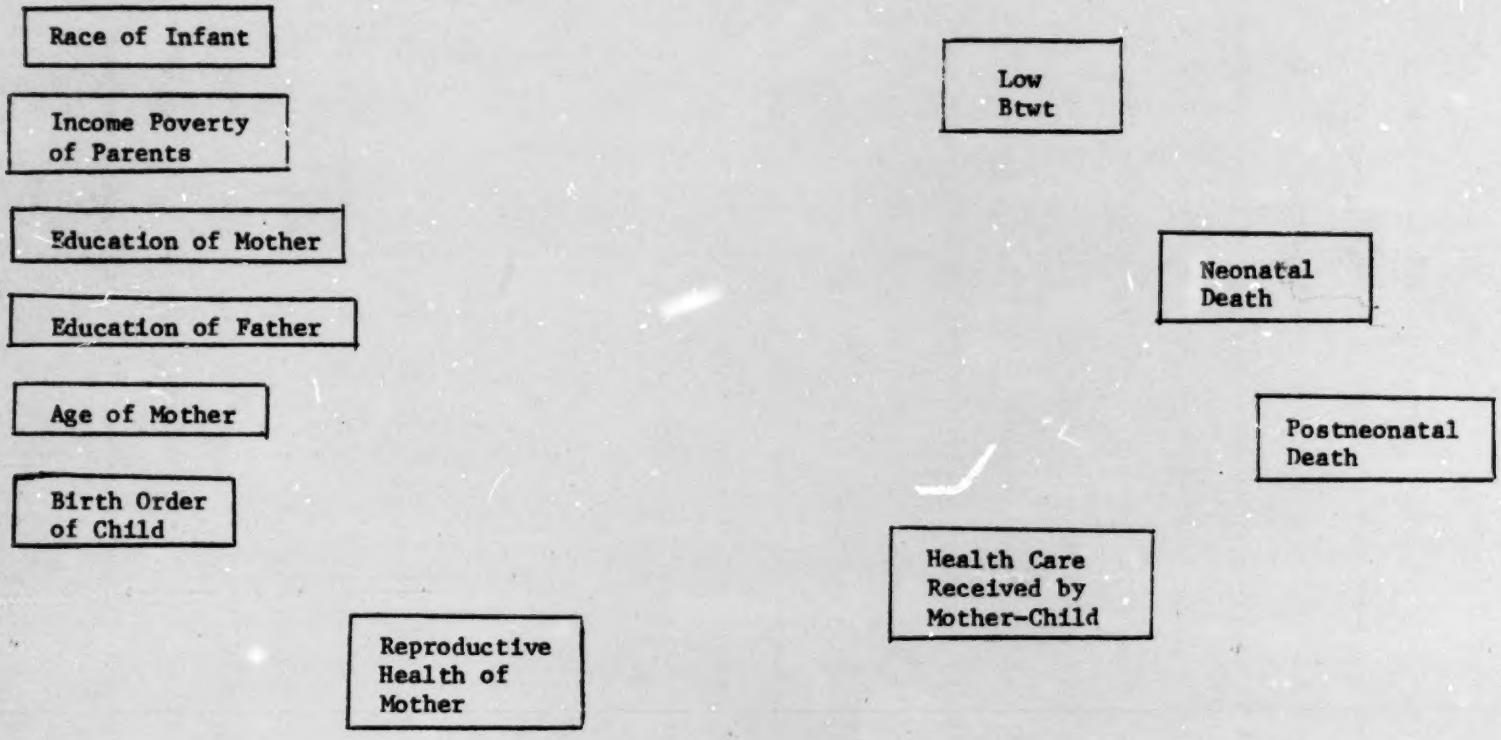

Figure 1. Recursive causal diagram of factors affecting neonatal and postneonatal infant mortality. Causal relationships between factors included in this design may be visualized as arrows between the boxes.

infant death, when operationalized, take on the status of intervening variables in the infant mortality process. No causal structure is being imposed upon the variables listed contemporaneously with the stratification factors. Although variables such as birth order and age of the mother might be seen as causally related to the stratification position of the parents, reciprocal paths of these sorts are also quite plausible, and it will not be attempted here to sort out these divergent sets of relationships. Because, as we have already noted, the variables included in the present analysis do not include all possible routes by which stratification position of the parents may influence infant death, our estimates of the impact of poverty and race upon infant mortality may perhaps be seen better as reduced form estimates of these effects, holding constant other factors in the model. In particular, the prenatal care received by the mother is not included in the present data set. However, to the extent that poverty and race relate at all to infant death, via whatever mechanisms, the present model should give reasonable estimates of their impact.

3. THE DATA

Between 1964 and 1965 the National Center for Health Statistics (NCHS) undertook the National Natality and the National Infant Mortality Surveys. These surveys were designed to obtain information not normally found on birth and death certificates—in particular, questions about the socioeconomic characteristics of the infants' parents were asked. Very similar questionnaires were used for both the birth and infant death

surveys, thus estimates of infant death rates for the United States as a whole for a variety of categorizations were made possible. Only deaths to legitimate infants were included in the study.

The National Natality Survey (NNS) of 1964-66 was based upon a probability sample of 1 out of every 1,000 birth certificates received by NCHS; all states of the United States are included in this sample. The National Infant Mortality Survey (NIMS) was based upon a probability sample of 1 out of every 11 deaths under one year of age included in the Current Mortality Sample, which is itself a systematic sample of 1 out of every 10 death certificates received by NCHS each month. All states are included in this sample, also (NCHS 1972, Appendix I). Since the 1950 birth registration test showed that 98% of live births were being registered, and since infant deaths are believed to have comparable rates of registration, the effects of underregistration in these samples is no longer considered a problem, at least with respect to national rates (National Office of Vital Statistics 1950, Volume I, Chapter 6).

Information on respondents was obtained from a variety of sources: birth certificates (NNS), death certificates (NIMS), hospital records (NIMS), as well as from the questionnaires themselves (both NNS and NIMS) The questionnaires were usually completed by the mother, but in rare instances other family members (NCHS 1972, p. 2). The questionnaires were initially mailed to respondents; follow-up procedures were used, including remailings as well as personal interviews, if no response occurred within two to three weeks. "For the 2,160 legitimate infant deaths in the 1964-1966 NIMS, the response rate was 88%. For the 10,395 legitimate births in the 1964-1966 NNS, the response rate was 89%" (NCHS 1972, p. 46).

Individual respondents were assigned a post-stratification weight based upon the representativeness of the sample within categories for which information on all United States births or infant deaths were available (e.g., a cross-classification of all U.S. births and infant deaths during 1964-66 by the variables of age of mother, race, live birth order, and legitimacy status is available from Vital Statistics of the United States 1964-66). Thus the statistics estimated in the present research from tables based upon these post-stratification weights should be more representative of the population of births and infant deaths than would be expected with a random sample alone (NCHS 1972, p. 50).

The question of how to treat nonresponse in a survey is certainly difficult to answer. Two techniques were used by the administrators of the NNS and NIMS surveys. One technique dealt with "item nonresponse," or the fact that a questionnaire was returned, but information on an item was missing. Follow-ups were attempted in order to supply the missing information; if this failed, responses were imputed. The method of imputation depended upon the question: for example, family income, which had the highest unit nonresponses in both the NNS and NIMS surveys (2.5% and 7.3%, respectively), was generally imputed on the basis of father's education and the household listing, although each case was considered individually and other information was used. Given this information, another questionnaire with similar characteristics was selected at random and the responses on this record were given to the item in question. Item nonresponse rates for most items on both surveys were less than 1% (NCHS 1972, pp. 49-50).

The effect of such item nonresponse upon relationships between variables estimated from the data is difficult to ascertain. Blau and Duncan, however, estimate that item nonresponse in their survey of occupational changes in a generation (sample size of approximately 20,000; nonresponse rates of 4.6% and 11.7% on the questions studies), and the subsequent bias in the sample which results, leads to "errors in correlation coefficients.. . . . perhaps of about the same order of magnitude as the sampling error of these coefficients" 1967, Appendix F). This observation gives just another reason to be conservative in our interpretation of the statistical significance of our estimated effects.

"Unit nonresponse" refers to the fact that no questionnaire was returned. Even though a questionnaire was not returned, information for this respondent was still available from the birth or death certificates, which included questions concerning the mother's race and age, as well as live birth order. Imputation for unit nonresponse was made based upon these known characteristics in a manner similar to that used for item nonresponse. Since we know the records which were thus imputed, we may estimate the effects of such imputation upon the relationships being studied by dropping these respondents from the sample and re-estimating the model under consideration.

Methods of Mortality Analysis

Regression analysis has proven to be an exceedingly useful and accepted tool of analysis in many areas of nonexperimental social science research. Rather than merely documenting differentials in income, for example, status attainment researchers have constructed rather complex causal models which test hypotheses concerning the reasons individuals

attain different levels of income (Duncan 1968). The study of mortality from such a perspective has faced a number of obstacles, however. The main problems have been due to the nature of the dichotomous dependent variable in mortality analysis; this fact gives rise to a problem of unavoidable heteroscedastic errors in regression which results in inefficiencies of estimation and problems in making statistical inferences, and the model itself allows the estimation of probabilities outside the range of 0 - 1 (Goldberger 1964; Nerlove and Press 1973). Given the relatively small numbers of deaths involved in most mortality studies, researchers in the past have thus treated regression statistics with caution.

Fortunately, however, solutions to the above-mentioned problems have been developed in the statistical social science literature. Discrete multivariate logistic, or modified multiple regression models, in large part developed and exposited by Yvonne M. M. Bishop (Bishop and Mosteller 1969; Bishop; Fienberg, and Holland 1975) and Leo A. Goodman (1972; 1973a; 1973b) are quite well suited to the mortality analysis being undertaken here.

Quantitative models of the following form will thus be used in the present research as a means of specifying and estimating the relationships between variables of interest:

$$E(\text{death rate}_{ij \ldots n}) = \gamma\gamma_i^A \gamma_j^B \ldots \gamma_n^N. \qquad (1)$$

This formula states that the expected death rate for cell ij . . . n of the cross-classification table can be written as a product of the γs. Furthermore, it is a property of the model that $\prod_k \gamma_k^K = 1.0$, for K=A,B, . . . , N. The i,j, . . . , n refers to levels of A,B . . . , N. We may also take the natural logarithms of both sides of formula (1) and thus obtain the

equivalent expression

$$\lg E(\text{death rate}_{ij\ldots n}) = B + B_i^A + B_j^B + \ldots + B_n^N, \qquad (2)$$

where $B=\lg\gamma$, $B_i^A = \lg\gamma_i^A$, $B_j^B = \lg\gamma_j^B$, . . . , $B_n^N = \lg\gamma_n^N$. Furthermore, it is a property of this model that $\sum_k B_k^K = 0.0$.

Formula (2) may be recognized as being analogous to a linear, additive analysis of variance model (Scheffé 1959) containing terms for the "main effects" of the variables of interest, the B_n^N, as well as something analogous to a "grand mean," the B. Terms analogous to "interaction effects" in analysis of variance may also be added to formula (2), e.g., B_{ij}^{AB}.

Maximum likelihood estimates of the γs (or equivalently, of the Bs) can be obtained under a variety of sampling schemes using programs such as ECTA.[2] The fact that such estimates may be made under a variety of sampling situations is fortuitous for the present research; as was noted in the section describing the data of this project, probability samples of 1 out of every 1,000 births and 1 out of every 110 deaths were selected. It can be shown (Bishop, Fienberg, and Holland 1975, Chapter 3) that one need not differentially weight these samples in order to obtain maximum likelihood estimates of the B_n^N (or γ_n^N) parameters. The $B = \lg\gamma$ parameter estimates will be affected, although maximum likelihood estimates of B may be obtained by differentially weighting the samples. The great virtue in not having to differentially inflate observations is that no corrections for degrees of freedom need be made when doing statistical tests. Statistical tests themselves are based upon χ^2 distributed statistics.

One may interpret the effect parameters estimated under a model such as that given above in a variety of ways. Some useful approaches are the following, all of which will be used throughout the present research in order to facilitate the interpretation of results:

1. The relative sizes of the effect parameters indicate the relative influence upon the dependent variable of the different independent variables in the model, or their interactions. (This fact should be rather obvious.)

2. It should be noted that the functional form of model (1) is multiplicative. Thus, in formula (1), the relative effect upon the expected death rate of being in the first category of variable A versus being in the second category of variable A depends not only upon the estimated values of γ_1^A and γ_2^A, but also upon the level of the product $\gamma_j^B \ldots \gamma_n^N$. An example may make this property of the model clearer. For simplicity, let variable A have two categories: γ_1^A = being in income poverty, γ_2^A = not being in income poverty. Further, let our sample estimates for these coefficients be $\hat{\gamma}_1^A = 1.2$ and $\hat{\gamma}_2^A = 1/1.2$. Then, the effect of being in income poverty upon the expected death rate is to multiply the product $\hat{P} = \hat{\gamma}_j^B \ldots \hat{\gamma}_n^N$ by 1.2, while the effect of not being in income poverty is to multiply this product by 1/1.2 (holding constant the effects of all other factors). If $\hat{P}$ is relatively small (i.e., if the other characteristics of the child do not place it in very great risk), the effect of $\hat{\gamma}_1^A$ upon the expected death rate will be relatively small. If, however, the child's other characteristics place it in extreme risk (e.g., mother has experienced previous pregnancy

loss, no prenatal care, etc.), the effect of $\hat{\gamma}_1^A$ upon the expected death rate will be considerably larger in absolute terms.

This functional form makes much sense in mortality analysis: one intuitively expects that as an infant is subject to greater and greater risks, factors become relatively more crucial in producing higher absolute rates of death. This multiplicative form has been used quite extensively in the econometric literature; production function applications are typical. Furthermore, by adding interactions to the model, one can test the appropriateness of this functional form.

3. One may also speak of the relative effects of the parameters estimated in terms of percentage changes in the dependent variable. For example, the effect of $\hat{\gamma}_1^A$ is to multiply $\hat{P}$ by 1.2; conversely, the effect of $\hat{\gamma}_2^A$ is to multiply $\hat{P}$ by 1/1.2. Thus, the relative effect of $\hat{\gamma}_1^A$ versus $\hat{\gamma}_2^A$ is to increase the expected death rate under the given model by a factor of $(1.2)^2 = 1.44$. Taking condition 2 as a base (not being in poverty), one can say that the effect of the poverty condition, net of the other variables in the model, is to increase the death rate 44%. Similarly, one can obtain a comparable figure from the log form (2) by taking twice the difference between $\hat{B}_1^A$ and $\hat{B}_2^A$ and taking to the e^{th} power. This sort of interpretation of the estimated effects is equivalent to the epidemiological notion of relative risk (MacMahon, Pugh, and Ipsen 1960, p. 229).

Variables Used in Analysis

Two varieties of infant death were analyzed separately: 1) neonatal mortality (NEON), or those deaths to infants which occur during the first 28 days after birth, and 2) postneonatal mortality (POST), or those

deaths to infants which occur during months 2-12 after birth.[3] The substantive rationale for this breakdown has already been discussed.

Four aspects of the infant's parents' stratification position are operationalized. First, as was previously noted, one aspect of the theory being tested relates to the parents being in a position of extreme income poverty. This concept was operationalized along income poverty guidelines provided by the Community Services Administration (CSA), formerly the Office of Economic Opportunity (Community Services Administration 1975). Parents are thus classified as either in or out of poverty (POV). These guidelines were adjusted to take into account family size, and thus to provide a rather accurate representation of a family's true ability to afford goods and services. The guidelines were also adjusted to reflect increases in consumer prices over the period 1965-74, as charted in the Consumer Price Index.

Applying the original guidelines as set by the CSA led to approximately 35% of the population under study (and approximately 70% of the black population) classified as being in poverty. The guidelines were subsequently revised downward so that approximately 20% of the sample is now classified as being in poverty, this breakdown being more in line with our theoretical concerns to look at those in extreme poverty. The guidelines are detailed in Appendix A.

Two other dichotomous attributes of the infant's parents were used to classify them as lying at one extreme of the stratification system: both low education of mother (less than nine years completed education) and low education of father (less than nine years completed education)

are used (EDM and EDF, respectively). Possession of such low educational attainment may be seen as indicative of a variety of nonfinancial characteristics of the family environment, such as knowledge of health practices, or of a more general cultural isolation.

Whether the respondent was black or white (RACE) was used to define another aspect of stratification. Small numbers of cases precluded the inclusion of other racial-ethnic groups.

Age of mother (AGE) was defined by four categories: 20, 20-24, 25-34, and 35+. Birth order of the child (NBTH) was likewise broken down into four categories: 1st, 2nd, 3rd, and 4th or higher birth. Whether or not the mother experienced a previous pregnancy loss (fetal death or miscarriage) or an infant death is represented by the variable (PRPG).

Two variables relating to the health care experience of the mother (and hence the infant) are included in the present analysis: these concern whether or not health insurance for physician and hospital care is possessed by the parents (INS), as well as whether or not the baby was delivered in a hospital (HOSP). The effect of (INS) upon infant death will indicate to us the extent to which the ability to finance health care, as reflected in the possession of health insurance, may influence the infant death process. Not being born in a hospital may certainly have detrimental effects upon an infant's life. Besides indicating that no intensive care facilities would be available in case of emergency, however, this variable may also indicate a more general lack of health care for the infant; such a fact would be indicated if (HOSP) is significantly associated with both neonatal and postneonatal mortality.

Finally, the birthweight (BTWT) of the infant was coded as either less than or greater than 2500 grams, the commonly accepted definition of prematurity.

As was noted previously, conceptualizing the infant mortality process as a causally ordered sequence leads to the specification of a variety of dependent variables; some of these may be considered the outcome variables of greatest interest, such as neonatal mortality and postneonatal mortality, while others, such as the health care received by mother and infant, may be considered intervening variables. Operationalizing this view of the infant mortality process in terms of the variables defined above results in the following sets of functional relationships:

(1) PRPG = f(POV, EDM, EDF, AGE, NBTH)

(2) INS = f(" " , PRPG)

(3) BTWT = f(" " , INS)

(4) HOSP = f(" " , BTWT)

(5) NEON = f(" " , HOSP)

(6) POST = f(" ")

One point of possible confusion should be commented upon: while in the conceptual diagram presented in Figure 1, health care was located causally prior to the infant's birthweight, here in (4) we are acknowledging the fact that low birthweight, and its attendant hazards, may lead to the hospitalization of an infant, rather than vice versa.

Each of the dependent variables in (1) to (6) contains two categories. Hence, each of the relationships indicated may be translated into equations of the form indicated in equations (1) or (2) on pages 20 and 21. The dependent variable in all these instances may be described as the expected odds of being in one category of the dependent variable versus

being in the other category, conditional upon given values of the independent variables. In the case of NEON and POST, we are talking thus in terms of expected death rates, since the two categories correspond to deaths and births. For the other dependent variables, characterized by equations (1) to (4) above, it should be recognized that none of the recorded deaths are employed as data in specifying or estimating these equations--no one has yet died. Thus, the sample for these equations consists of all births, while the sample for the equations characterizing NEON and POST consists of all births and deaths.

In the case of BTWT, it is useful to think of the expected odds of an infant being less than 2501 gm at birth, versus being greater than 2500 gm as being the "prematurity rate". One question which might arise at this point concerns the use of a dichotomous measure of low birthweight as an indicator of fetal development, rather than a continuous measure, such as the infants birthweight in grams, or the gestation length in weeks of the infant. The use of birthweight rather than gestation length in the present research may be justified because of birthweight's closer connection to environmental factors; recent research has supported the view that birthweight is more severely affected by environment than is maturation (Gruenwald 1974; Gruenwald et al. 1967). The use of a dichotomous measure, rather than a simple linear one, is indicated by the fact that infant death rates are related to birthweight in a nonlinear fashion. For those infants weighing less than 2500 gm at birth, death rates are quite high; among those infants weighing more than 2500 gm, however, there is substantially less variation in the risk of death. Stated another way, a difference in birthweight of 200 gm means little, on the

average, if one is speaking of infants weighting 3500 and 3700 gm. However, this difference has significant implications for survival if one is referring to infants weighing 1500 and 1700 gm. Thus, the use of a dichotomous indicator of low birthweight should most adequately represent the indicated relationship.

Maximum likelihood estimates for equations of the form of equation (2) on page 21 (or, equivalently equation (1)) were made for the different dependent variables of the postulated recursive model. For white infants, these are presented in Figure 2 and Table 1. Figure 2 only includes coefficients which tested significantly different from zero at $\alpha = .05^4$. Significance tests for effects of interest were made according to two rules: 1) The full "additive" model was estimated first, thus assuming no higher order interactions, and main effects were then dropped one at a time, with the model re-estimated each time in order to provide χ^2 tests for the significance of the dropped coefficient. Such χ^2 tests, along with a table of significance levels, are presented in Table 1. 2) Tests for higher order interactions were made by adding interactive terms to the model derived above, and then testing for significant changes in χ^2 statistics.

A full model was run first, which included all the variables of interest described in the preceeding section, including race of the infant. A number of significant race interactions were found, indicating that the process of infant mortality operates significantly differently for whites and blacks. This constitutes a significant finding in itself: prior research based upon a Wisconsin sample, for example (Slesinger and Travis 1974) reported no significant race effects, although it

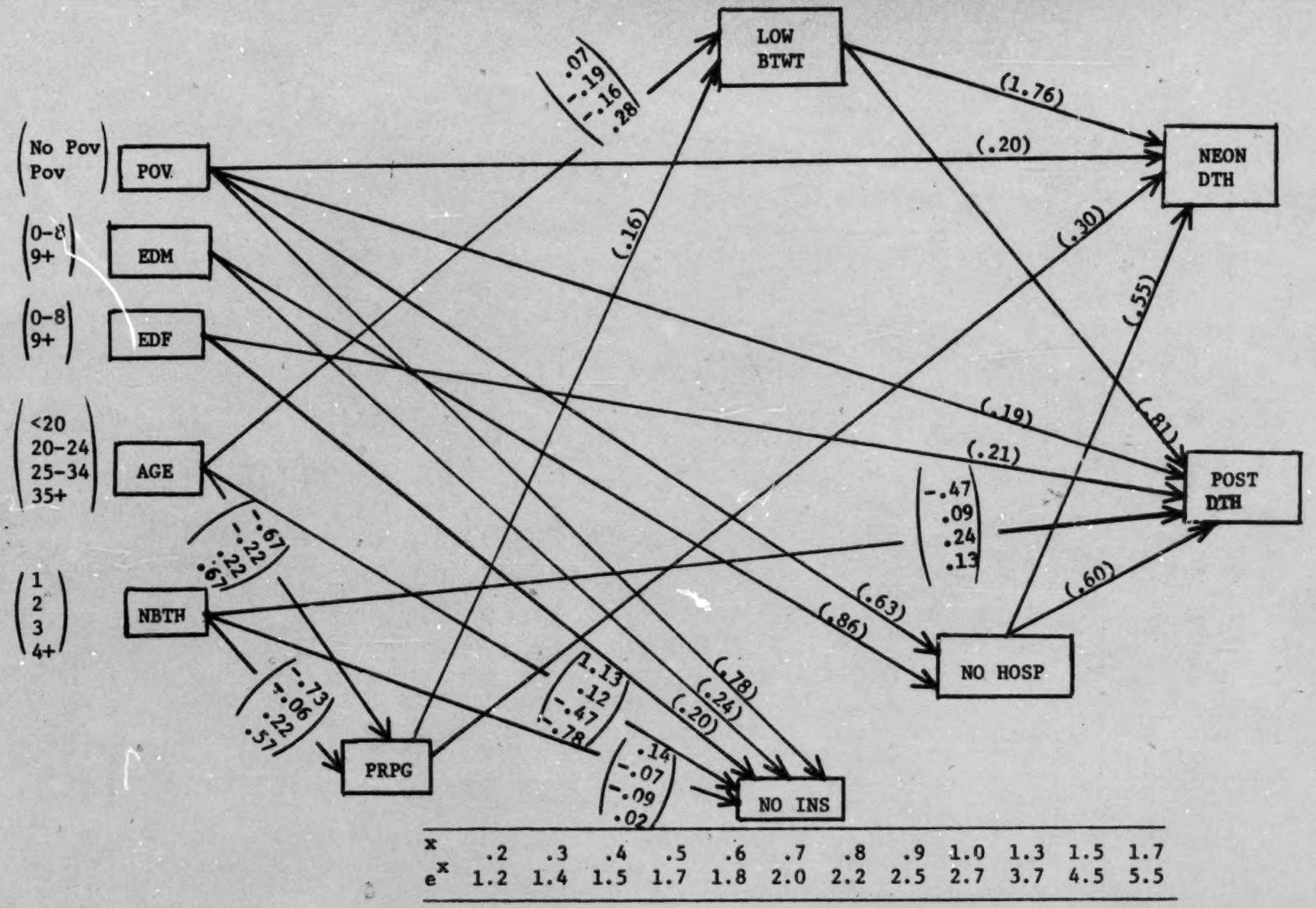

Figure 2: Factors affecting neonatal and postneonatal mortality among white, legitimate infants in the United States in 1964-1965

Figure 2--Continued

Note: N = 6,185 births, and 878 neonatal, 303 postneonatal deaths. All effects (or sets of effects) shown are significantly different from zero at $\alpha = .05$.

For dichotomous effects, one can interpret the direct effects in terms of relative increases in the death rate; e.g., the direct effect of POV upon the neonatal death rate is to increase it $e^{.40} = 1.49$ times over that experienced by infants not born in poverty (all other factors held constant).

Table 1

Coefficient Estimates: Factors Affecting Neonatal and Postneonatal Mortality, for White, Legitimate Births, 1964-1965 in the United States.

PREDETERMINED VARIABLES		DEPENDENT VARIABLES					
		PRPG	NO INS	LOW BTWT	NO HOSP	NEON DTH	POST DTH
No Pov	POV	.03	.78	.02	.63	.20	.19
Pov		(.41)	(324.72)**	(.06)	(11.79)**	(9.63)**	(5.44)*
0-8	EDM	.04	.24	.07	.86	.02	.15
9+		(.55)	(21.39)**	(.66)	(20.82)**	(.10)	(2.67)
0-8	EDF	.00	.20	.05	-.04	.02	.21
9+		(.00)	(20.23)**	(.45)	(.03)	(.09)	(6.72)**
20	AGE	-.67	1.13	.07	.33	.16	.40
20-24		-.22	.12	-.19	-.37	-.02	.06
25-34		.22	-.47	-.16	.06	-.10	-.14
35+		.67	-.78	.28	-.02	-.05	-.31
		(93.31)**	(256.71)**	(10.12)*	(1.85)	(2.30)	(7.43)
1	NBTH	-.73	.14	-.09	-.80	-.04	-.47
2		-.06	-.07	-.02	-.00	.12	.09
3		.22	-.09	-.00	.06	.02	.24
4+		.57	.02	.12	.75	-.09	.13
		(165.85)**	(9.30)*	(1.69)	(6.57)	(2.79)	(13.97)**
None	PRPG		-.04	.16	-.02	.30	.05
1+			(1.48)	(8.26)	(.01)	(34.86)**	(.46)
None	INS			.10	.33	-.07	.07
Some				(3.09)	(3.22)	(2.05)	(.94)
≤2500	BTWT				-.33	1.76	.81
2501+					(1.04)	(1,743.73)**	(112.68)**

Table 1—Continued

	PRPG	NO INS	LOW BTWT	NO HOSP	NEON DTH	POST DTH
(No Hosp / Hosp) HOSP					.55 (7.65)**	.60 (9.57)**
χ^2 (degrees of freedom) for equations	143(118)	254(245)	381(500)	127(1011)	631(2034)	522(2034)

Note: χ^2 tests are in parentheses. * = significant at .05; ** = significant at .01.

should be noted that the black population of Wisconsin is quite small. (Racial differences will be commented upon later.) We turn now to a more detailed analysis of findings with respect to the white population. The sample size for this population is large enough to make fairly reliable statements concerning the estimated effects. Comments concerning the black population will necessarily be more cautious, in consideration of the smaller sample involved.

In discussing the estimated models, some comments should first be made concerning the fit of the various estimated equations to the data.

With respect to the explained variance of these models, some misconceptions should be cleared up: researchers in the past using regression models to analyze infant mortality have noted with disdain the low R^2s produced by such models, the inference being that a low R^2 means the model has no explanatory power (see e.g., Kessner 1973, pp. 63-64). Two misunderstandings are involved here. First, because of necessarily heteroscedastic errors, the multiple R^2 statistic is not appropriate for making statistical decisions in dummy-dependent variable regression (Nerlove and Press 1973, p. 7). Secondly, in the sense of prediction, one should expect a low R^2 in mortality analysis because of the nature of the dependent variable. Given practically any set of attributes of a liveborn infant, for example, one's best guess as to whether it will live or die is usually that it will live. Infant mortality occurs in less than 1 of 50 infants, and given the nature of the dependent variable, prediction of individual death is thus quite difficult. What is important for our work is not that one can predict precisely who will die, but rather that one note which factors have the greatest influence in changing the probability or relative risk of death. Such inferences are made

through examination of structural parameters, conditional upon the fact that an adequate statistical model has been specified and fitted. The models used here are appropriate for the present data, and an adequate fit is achieved. Statistics analogous to R^2 have been developed for models similar to the one used here; an example is the concept of "expected mutual information" (Theil 1971, Section 12.8). We will not go into a discussion of this concept here, only to note that, as expected, the present models have fairly low predictability on an individual level basis.

4. CAUSAL ANTECEDENTS OF INFANT DEATH: THE WHITE POPULATION

From looking at Figure 2 it should be quite apparent that income poverty, as defined here, is a major antecedent of lack of health insurance among the white population of infant's parents. Controlling for the other factors in the model, poverty status leads to a five-fold increase ($e^{2(.78)} \simeq 5.0$) in the chances of a family not possessing health insurance over those families not in poverty. Likewise, poverty status of the family leads to a tripling of the odds of the infant not being born in a hospital over families not in poverty.

These findings support the well-accepted notion that access to health care in the United States is significantly structured by the stratification system. It is also of interest to note that the largest effects upon possession of health insurance are those associated with age of the mother--in particular, those mothers less than 20 years of age.

Let us digress for a moment and examine in detail the implications of these first estimated relationships. Specifically, although the reporting of relative risk is a useful measure of the strength of these relationships, we would also like to estimate the numbers of individuals implicated in these associations. In the present context, although the data demonstrate a quite strong partial effect of income poverty upon nonhospitalization of the mother at birth, it is a fact that only approximately 1.6% of all births during the study period did not occur in a hospital; correspondingly, it may be roughly estimated that 13,000 infants were not born in a hospital due to this poverty factor, which constitutes approximately .2% of the total infant population. (See Appendix B for a detailed discussion of this estimation procedure.)

No statistically significant direct effects of stratification factors upon birthweight were found. The fact of possessing some insurance for health care (INS), however, did exhibit an effect significant at the .10 level. The magnitude of this effect indicates that possession of insurance for maternal and infant health care leads to a 20% reduction in the relative risk of a mother having a low birthweight infant. This finding supports a theory that health care--and in particular, prenatal care--may be instrumental in lowering rates of prematurity, as the positive effect of prenatal care in reducing the risk of low birthweight has been convincingly documented elsewhere (Kessner et al. 1973; Gortmaker 1977).

Coefficients similar in sign, but smaller in magnitude likewise characterize the partial relationships of the other stratification

variables to BTWT; the statistical insignificance of these effects, however, preclude discussion of their role in the structuring of the risk of low birthweight. Needless to say, these estimated relationships are of small magnitude. In contrast, the differentials in risk associated with age (AGE) and the mother's previous pregnancy experience (PRPG) are large. Both of these latter findings are consistent with previous research (O'Sullivan et al. 1965) although, as was noted earlier, the observed relationship between young age of mother and low birthweight may reflect a relationship between low prepregnant weight of the mother and low birthweight of the infant, and this relationship in turn may also reflect a lack of prenatal care.

Determinants of Neonatal and Postneonatal Death

The largest estimated effects upon both neonatal and postneonatal mortality, as expected, are those associated with low birthweight of the newborn infant. The fact that our model demonstrates few significant relationships of low birthweight with antecedent stratification factors--with the exception of the intervening health insurance variable (INS)—tells us that although birthweight is quite an important factor in infant death, it is not an especially significant link in specifying the relationship of income poverty to infant death, at least in the white population. (We will later note the lack of a significant relationship in this respect in the black population, also.) This lack of substantial relationships between developmental factors in infant death (low birthweight) and income poverty should sensitize us even more to the possibility of other factors being implicated in class differentials,

including the influence of health care during birth and after the infant in born. This discussion is not meant to imply that cases of extreme nutritional deprivation in poor mothers do not exist, that such deprivation does not have harmful effects upon the development of the newborn, or that prenatal care has only a minimal impact upon BTWT. The current data is obviously inappropriate for answering these questions adequately. On the contrary, in fact, the evidence given here is consistent with such perspectives. However, the point is that the largest effects of income poverty do not operate via the fact of low birthweight.

The fact of nonhospitalization of the infant at time of birth is quite strongly associated with both neonatal and postneonatal death, as indicated in Figure 1. The fact that nonhospitalization exhibits equally large effects upon these two outcomes, however, tells us that more than the mere fact of nonhospitalization is probably being measured. This variable might be better indicative of a more general lack of any medical care. It is perhaps sobering to make some rough estimates of the impact of income poverty upon infant death via its association with nonhospitalization. It may be estimated that the excess neonatal death rate due to nonhospitalization is .0312. (Again, see Appendix B for a discussion of the estimation procedure used.) Multiplying this figure by the estimated number of NO HOSP infants due to their poverty status (13,000—derived above) gives us an estimated 370 deaths via this indirect route over the two year sample period. The fact that these coefficients are statistically significant, and our estimates of the number of deaths involved conservative, should convince the reader of the "realness" of this association,

even though the number of deaths involved would have little impact upon any national rates. This finding does, however, illustrate one of the extreme effects of the stratification system as it relates to the health care of infants and infant death.

Significantly, we may observe that parental lack of health insurance, as measured here, does not directly alter an infant's chances for survival, controlling for the other variables in the model. The reader should recall, however, that a significant effect of possession of health insurance upon low birthweight was found. Furthermore, having health insurance (as defined by INS) means only that some insurance was possessed by the parents, and thus real financial barriers to health care may still exist among those classified as having insurance; this variable may thus be measured with considerable error. Therefore, these results must be interpreted with caution, but the evidence certainly indicates that changes in the possession of health insurance in the United States would have a significant impact upon the health of the newborn population, although the "indirect" effects of poverty via this mechanism are small, compared with the direct effects of poverty discussed below.

Significant direct effects of income poverty level--both in the neonatal and in the postneonatal period--upon infant death were demonstrated. This finding is perhaps the most significant of the present analysis. The magnitude of the estimated coefficients indicates that being born into income poverty versus not increases an infant's risk of death by almost 50%--both in the neonatal and in the postneonatal period. We may again make a conservative estimate of the number of lives involved: for the neonatal period, this amounts to approximately 6,600 deaths;

during the postneonatal period, this amounts to roughly 2,100 deaths. Both of these estimates are for the period 1964-65. The infant mortality rate for the white population studied was 21.0 per 1,000 in 1964-65. If the poverty effects just discussed had not existed at that time, the rate would have been 19.6 per 1,000, not an insignificant decline.

5. STRATIFICATION, HEALTH CARE, AND INFANT DEATH: THE BLACK POPULATION

Extensive discussion and analysis has already been presented concerning the determinants of infant death, as defined by this study, among the white population of the United States as a whole in 1964-65. Now we turn to a discussion of estimates made for the black population of the United States during the same period. The reader may wish to review some of the previous analysis of data for the white population, although relevant contrasts will be pointed out when the present data for the black population is discussed.

With respect to the NCHS data, our analysis of the process of infant mortality among the black population, as contrasted with the white population, will necessarily be brief, due in large part to the fact that such a small sample of black births and deaths was obtained: 840 births, including 117 low birthweight infants, plus 206 neonatal and 105 postneonatal deaths constitute the study population of black infants. Our interpretation of statistics based upon these data will be necessarily cautious.

Structural coefficient estimates (of the log-linear, or additive form) are presented in Figure 3 and Table 2. With respect to the determination of antecedent conditions of infant death, there is little to distinguish

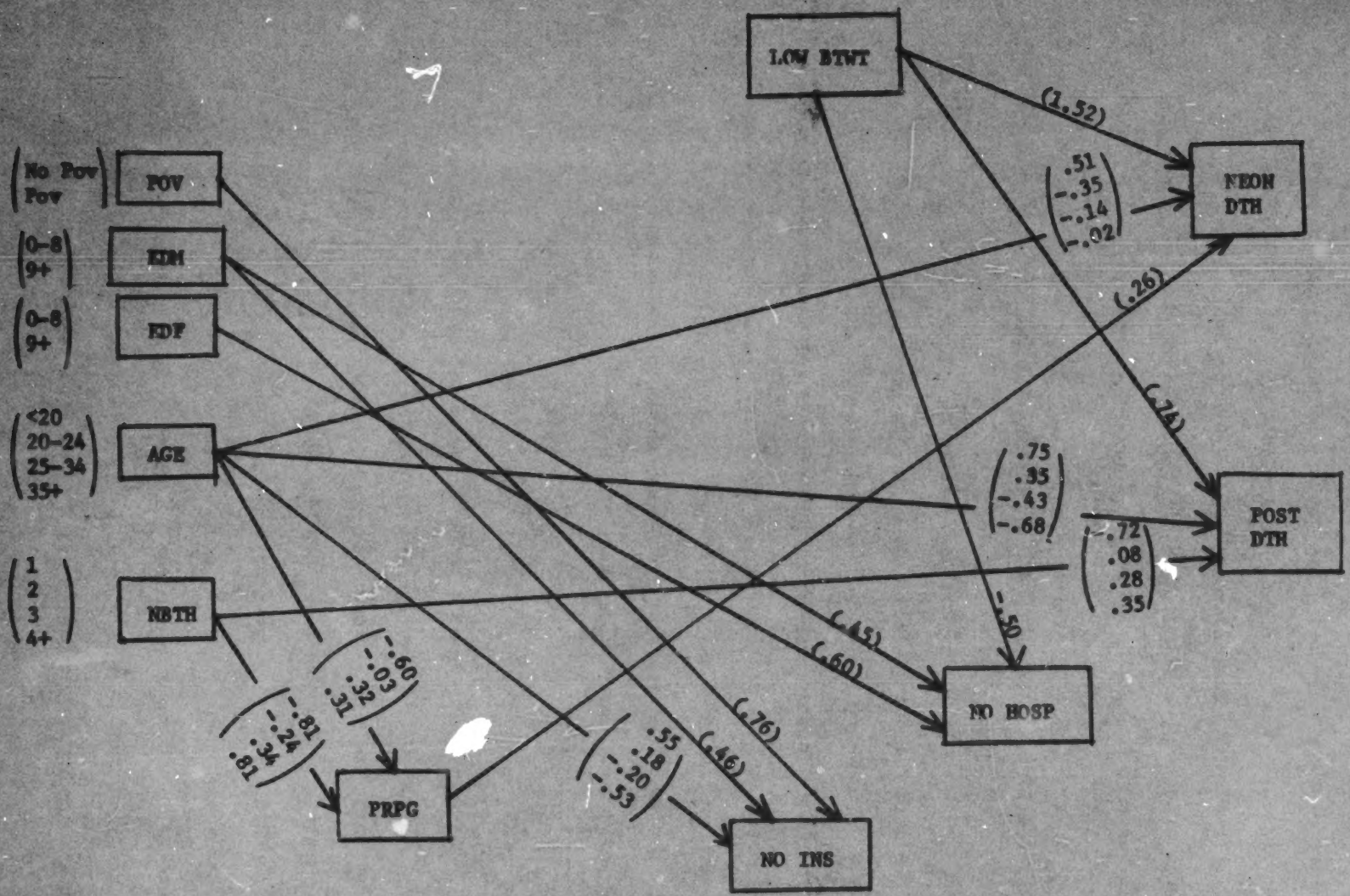

Figure 3: Factors affecting neonatal and postneonatal mortality among black, legitimate infants in the United States in 1964-1965.

N=840 births, and 206 neonatal, 105 postneonatal deaths. All effects (or sets of effects) shown significantly different from zero at $\alpha = .05$.

Table 2

Coefficient Estimates: Neonatal and Postneonatal Mortality, for Black, Legitimate Births, 1964-1965 in the United States

PREDETERMINED VARIABLES		DEPENDENT VARIABLES					
		PRPG	NO INS	LOW BTWT	NO HOSP	NEON DTH	POST DTH
No Pov Pov	POV	.02 (.04)	.76 (75.21)**	.06 (.24)	.35 (3.12)	-.16 (2.10)	.03 (.04)
0-8 9+	EDM	.20 (3.74)	.47 (15.84)**	.13 (.85)	.45 (8.84)**	.08 (.37)	.00 (.00)
0-8 9+	EDF	.13 (1.66)	.01 (.00)	-.14 (1.15)	.60 (13.65)**	-.03 (.05)	.18 (1.68)
20 20-24 25-34 35+	AGE	-.60 -.03 .32 .31 (8.68)*	.55 .18 -.20 -.53 (10.90)*	-.07 -.20 .01 .27 (1.74)	.10 -.14 .14 -.10 (.90)	.51 -.35 -.14 -.02 (8.45)*	.75 .35 -.43 -.68 (13.89)**
1 2 3 4+	NBTH	-.91 -.24 .34 .81 (39.62)**	.21 -.12 .11 -.21 (3.39)	.15 -.26 .35 -.24 (5.40)	-.15 -.15 .48 -.18 (2.97)	-.05 .10 -.05 .00 (.34)	-.72 .08 .28 .35 (8.86)*
None 1+	PRPG		-.05 (.30)	-.06 (.25)	.01 (.00)	.26 (6.44)*	-.01 (.01)
None Some	INS			.10 (.68)	.35 (3.71)	-.15 (2.06)	-.10 (.62)
≤2500 2501+	BTWT				-.50 (4.68)*	1.52 (290.39)**	.74 (38.21)**

Table 2—Continued

	PRPG	NO INS	LOW BTWT	NO HOSP	[illegible] DTH	POST DTH
No Hosp Hosp HOSP					.31 (3.26)	.13 (.46)
χ^2 (degrees of freedom) for equations	118(118)	172(245)	190(500)	141(1011)	366(2034)	306(2034)

Note: χ^2 tests are in parentheses. * = significant at $\alpha = .05$; ** = significant at $\alpha = .01$.

the white from the black process of infant mortality; in other words, the estimated coefficients for the two populations tend to be quite similar. (It should be recognized that one may accurately compare estimated coefficients from these multivariate logistic models across populations in the same manner that one would compare unstandardized regression coefficients.)

In the determination of neonatal and postneonatal mortality, some significant differences do occur. Quite large and statistically significant effects of age of mother (AGE) which directly affect NEON and POST are found in the black population; and these effects are statistically insignificant in the corresponding white equations. (Later, tests for differences between these coefficients will be given.) No explanation for these differences will be tested at this time, although this evidence is certainly indicative of the need for improved care among this young segment of the black population. Neonatal death rates for infants born to black mothers less than 20 years old are five times greater than the rates for those infants born to mothers aged 20-24, making adjustment for the effects of other factors included in our design. Other research by the author using data from New York City suggests that this relationship is due to a lack of prenatal care among young black mothers (Cortmaker 1977, Chapter VI).

No significant relationships between aspects of the stratification system, and in particular, the families' poverty status and infant mortality were found to exist in the black population, as opposed to the white population, where significant and consistently positive effects

were observed. Because of the small sample size involved, and hence the low power of our statistical models to detect small effects, however, I do not take this evidence as definitive of significant differences in the process of infant mortality between the two populations. Further research into this question is needed.

Finally, we should note that low birthweight has less effect upon neonatal death in the black as opposed to the white population. (Tests for the statistical significance of this difference will be presented shortly.) Analyses discussed below, however, will demonstrate that this difference contributes little toward explaining black-white differentials in infant mortality.

Given this overview of process differentials by race, we will now pursue an exercise calculated to estimate the extent to which racial differentials in infant mortality may be related to two distinct sources: 1) differences between the two populations in the distribution of infants over categories of variables, i.e., do black mothers tend to be younger, and does this fact account for some measured black-white differences; and 2) differences in process--these differences may be operationalized as "interaction effects"—which indicate that the process of infant mortality is significantly different (in a statistical sense) between the races. For example, we have noted above that low birthweight has less effect upon neonatal mortality in the black population than it does in the white population; we would further like to know how much of the racial differential in neonatal mortality may be attributed to this fact.

The contributions of these factors to observed infant mortality differentials between the races were estimated in the following manner:

the two data sets used for analyses within the black and white populations were combined, and an additional variable indicating race (RACE) was added to the design. Models predicting neonatal and postneonatal mortality were then estimated while selected factors were sequentially included in the design; all of these models included the RACE variable. Estimates of the RACE coefficient from these models thus demonstrate the effects of race as different aspects of the design are successively controlled. The factors which are successively included in these models consist of the following:

1. Demographic factors: AGE, NBTH
2. Stratification factors: POV, EDM, EDF
3. Health factors: PRPR, INS, HOSP
4. Birthweight: BTWT

The meaning of the "race" effect in these models changes, of course, as successive factors are included; essentially, this effect is meant to indicate the impact of all factors which are associated with race, which have not been controlled for in the given model. These questions of interpretation should become clear as we proceed to an examination of the evidence.

Table 3 below gives a list of relative risks which indicate the relative size of black versus white rates of neonatal and postneonatal mortality, as successive sets of factors are controlled in the given model. Thus, the observed neonatal and postneonatal mortality rate differentials for the black and white populations of 1964-65 in the United States may be characterized as "base relatives" of 1.73 and 2.55; in

Table 3

Relatives: Black versus White Rates, Controlling Successively for Demographic Factors, Stratification Factors, Health Factors and Birthweight in the United States, 1964-1965

	Neonatal death	Postneonatal death
Base relative	1.73	2.55
With demographic factors	1.62 (28.94)**	2.18 (36.58)**
With demographic and stratification factors	1.49 (17.88)**	1.75 (16.49)**
With demographic, stratification, and health factors	1.38 (11.26)**	1.65 (12.85)**
With demographic, stratification, health factors, and birthweight	.94 (.20)	1.42 (6.24)*
With all "additive" effects plus race x hospital interaction		.84
With all "additive" effects plus race x poverty and race x birthweight interactions	.94	

Note: * = significant at .05; ** = significant at .01.

other words, the observed black neonatal mortality rate was 1.73 times higher than the white rate, and the black postneonatal rate was 2.55 times higher than the corresponding white rate. We have already mentioned the relationship of these estimated "relatives" to estimates made from the multivariate logistic model (in the present case, estimates of the RACE coefficient are used to derive relatives).

If we control for differences in the distribution of demographic factors between the racial populations, these relatives are reduced to 1.62 and 2.18, respectively. The reader should be aware of the fact that these calculations assume (at least temporarily) that the process whereby these demographic differences are translated into mortality outcomes is the same for the two racial populations; furthermore, the estimated coefficients which define this common process are some weighted average of the coefficient estimates which define the separate black and white processes. Because our analysis has thus far unveiled few significant differences between the two populations, this assumption should not lead to large biases. Once all factors have been included in the design, however, we will specifically test for the presence of interaction effects between the races, and include such factors when appropriate.

Significant reductions in the estimated relatives may be noted as stratification and health factors are added to the design, giving a crude indication of their effect in accounting for racial differences in infant mortality. Statistically significant relatives of 1.38 and 1.65 still remain, however, when demographic, stratification, and health factors have been equalized. The importance of birthweight in accounting for differentials in neonatal mortality may thus be demonstrated: when

birthweight (BTWT) is added to the design, no significant racial differential in neonatal mortality remains. Furthermore, the addition of race x poverty and race x birthweight interactions does not change the magnitude nor insignificance of this residual race effect upon neonatal mortality. A complete table listing the statistical significance of all race x (variable) interactions is given in Table 4.

The fact that differences in demographic, stratification, and health factors, as defined here, may account for a change in relatives from 1.73 to 1.38 indicates the significant impact these variables may have in producing differentials in neonatal mortality. Furthermore, we should note that most of this "explained" difference may be attributed to differences in the distributions of these variables, and not to differences in process. These comments must be tempered, however, with a recognition of the small black sample employed in this exercise. Furthermore, the determination of such a high incidence of low birthweight babies in the black population for the most part remains unexplained, and this fact demands further study.

Differences in postneonatal death rates in the national data, as expected, may be significantly reduced by controlling for stratification, demographic, and health factor differences between the races. Quite a large (1.42) postneonatal relative remains after birthweight has been included in the analysis, but this effect is also reduced to unity when a statistically significant race x hospitalization interaction is included which indicates that non-hospitalization does not have a negative effect for blacks—as opposed to whites. We can offer no adequate substantive explanation for this interaction at this time.

Table 4

Race Interactions

Significance Tests: Individual Interactions and χ^2 tests

Race Interaction With:	Dependent Variable: Neonatal	Postneonatal
Pov	9.68** (1)	4.16* (1)
Edm	.33 (1)	4.77* (1)
Edf	1.15 (1)	3.52 (1)
Age	3.05 (3)	4.35 (3)
Nbth	.56 (3)	1.36 (3)
Prpg	.13 (1)	.80 (1)
Insl	2.44 (1)	3.38 (1)
Btwt	3.95* (1)	.36 (1)
Hosp	1.35 (1)	6.39* (1)

Coefficient Estimates: Final Model

Neonatal Mortality

Pov	Edm	Race	Edf	Age	Nbth	Prpg	Insl	Btwt	Hosp	Pov Race	Btwt Race
.03	.05	-.01	.00	.24	-.05	.29	-.09	1.64	.41	-.18	-.11
				-.09	.12					9.91** (1)	4.18* (1)
				-.11	.00						
				-.05	[illegible]						

Postneonatal Mortality

										Hosp Race
.15	.10	-.09	.20	.50	-.53	.03	.03	.79	.34	-.31
				.12	.08					6.39* (1)
				-.22	.27					
				-.40	.19					

Note: Neonatal mortality among the population of legitimate black and white infants in the United States, 1964-1965 (NCHS). Individual significance tests for race interactions, and coefficient estimates for "final" model which includes significant interactions. See text for discussion of testing procedure.

It is instructive to compare the estimates of relative risk reported here for black and white infants in the United States in 1964-65, with a similar decomposition performed with data from the state of Wisconsin in 1969 (Slesinger and Travis 1975). Some substantial differences in design may be noted: the following list roughly details the variables used in both studies:

NCHS (1964-65)	Wisconsin (1969)
Education of Mother	Education of Mother
Birth Order	Birth Order
Age of Mother	Age of Mother
Birthweight	Birthweight
Education of Father	Illegitimacy Status
Poverty Status	Residence (in or out of SMSA)
Insurance Coverage	
Hospitalization at Birth	
Neonatal Mortality	Infant Mortality
Postneonatal Mortality	

Further differences may be detailed with respect to the definition and categorization of the different variables.

In spite of these design differences, and in spite of the fact that few blacks live in Wisconsin and that Slesinger and Travis did not separately analyze neonatal and postneonatal mortality, somewhat similar estimates emerged from the two studies. For neonatal mortality, for example, the NCHS data provides an estimated racial relative of 1.38 when all factors but birthweight have been controlled; the corresponding relative for postneonatal mortality is 1.65. The Slesinger-Travis study, in contrast, produced a relative of 1.37 for infant mortality when all background factors but birthweight were included in the model.

When birthweight is added to the two models, the estimated racial relative for infant mortality drops to .97 (Travis and Slesinger 1975, Table 9), compared to relative estimates of .94 and 1.42 with the NCHS

data; addition of the aforementioned race x hospitalization interaction changes this latter estimated relative from 1.42 to .84.

A complete listing of significance tests for racial differences in process (interactions with the variable RACE) is given in Table 4. These are individual tests for interactions, which were calculated by adding the indicated interaction to the full additive model, and subsequently checking for a significant improvement in fit by comparing χ^2 statistics. The interactions which proved significant via this procedure were all added to the neonatal or postneonatal model, and were then dropped one-by-one as significance tests were made. In this manner, the three significant interactions included in the decomposition in Table 3 were chosen. The coefficients estimated for these final equations are given in the bottom half of Table 4.

In summation, our analyses of these national data have indicated the extent to which social and economic factors may account for racial differences in neonatal and postneonatal mortality; significant reductions in these differences are found when the factors are equalized, but large differences in birthweight distributions remain to be explained. These findings are not startling; in fact, they are quite consistent with previous research which has identified birthweight differences as the largest contributor to black-white infant mortality differentials. (Shapiro, Schlesinger, and Nesbitt 1968, pp. 53, 84; Susser, Marolla, and Fleiss 1972; Habicht et al. 1974; Slesinger and Travis 1975). Our problem at this point has thus become one of attempting to explain the causes of these different birthweight distributions; and furthermore, to

hopefully do this with reference to aspects of the social world which may conceivably be changed in the future; we have already referred to the fact that improved prenatal care constitutes one efficacious factor which can be changed to positively influence infant birthweight.

6. SUMMARY AND CONCLUSIONS

Our analyses of infant mortality differentials in the United States have confirmed a number of commonly stated beliefs: within the white population of legitimate births in 1964-65 in the United States, poverty status of the family is found to be associated with substantial differences in both neonatal and postneonatal mortality. After adjustments have been made for the effects of the variables education of mother, education of father, age of mother, birth order of the child, the mother's previous pregnancy experience, health insurance held by the family, birthweight of the infant, and whether the child was hospitalized at the time of birth the fact of income poverty is found to be associated with a relative risk of both neonatal and postneonatal death which is almost 50% greater than that experienced by infants not born in poverty. A conservative estimate of the number of lives involved may be given: if the poverty effects just described did not exist, approximately 6,600 infants during the neonatal period, and 2,100 infants during the postneonatal period would have survived.

Another significant aspect of these findings is the fact that these poverty effects are independent of the birthweight of the infant. A small effect of possession of health insurance (which is itself strongly influenced by the fact of poverty) upon low birthweight of the infant, however, was estimated, and may be interpreted as an

indication of the effects of prenatal care, since prenatal care has been found to exert a substantial negative effect upon infant mortality, mainly via its influence in reducing the risk of low birthweight (Gortmaker 1977)). Because prenatal care may be seen as perhaps the major health care influence upon the newborn, these results tend to support a common observation of physicians involved in the delivery of care to disadvantaged populations (Bennett 1976): increased access to health care can make a difference (the health insurance-prenatal care- effect upon birthweight), but poverty still, in a variety of other ways, tends to exert a substantial impact upon the well-being of an infant population. Thus, although our analyses have not been able to specify the medically oriented components of these poverty effects, we certainly must dismiss the notion that the changing nature of infant mortality has made poverty irrelevant to the health of infants in the contemporary United States.

Similar analyses were made for a smaller sample of black legitimate infants born in the United States in 1964-65. Few substantial differences in the process of infant mortality—as defined by the estimated structural equation coefficients--were found, although infants born to young black mothers experience particularly high mortality, and thus improved health care for this high-risk population is certainly needed.

Approximately half of the differences noted in the black-white infant death rates, both neonatal and postneonatal, may be related to differences in demographic, stratification, and background health factors between the two populations. A major conclusion, however, consistent with previous research, is that most of the remaining difference in infant mortality between the races may be attributed to differences in birthweight distributions. Improved prenatal care among the black population is suggested as one efficacious form of health care which could help to reduce this disparity.

It might appear to some readers that survey research of this nature always comes too late: 1964 and 1965 are already twelve years in the past, and the relevance of these data might seem to run the risk of being obscured by all of the social changes which have taken place during the ensuing decade. Many social problems, however, have a way of staying with us, and infant mortality differentials, as well as their reproduction via a stratified system of personal and health care resources, very likely constitutes one such problem. Thus, although infant mortality has been dropping steadily in the United States, it may be noted that black-white differentials remain quite substantial: in 1974, black infants experienced a force of mortality equivalent to that experienced by white infants in 1953 (National Office of Vital Statistics annual volumes 1975; 1954).

Furthermore, despite major government programs to eliminate poverty ("The War on Poverty"), and to more equitably distribute health care to mothers and children (Medicaid, Maternal and Infant Health Programs), differential distributions of these resources persist. Recent data on changes in income poverty in the United States, for example, illustrates this point well: in 1965 "25.5% of U.S. households were poor before taking account of government transfers. Over the whole period from 1965 to 1972, the incidence of household pretransfer poverty dropped by less than one percentage point" (Plotnick and Skidmore 1975, p. 170). Taking into account government transfers (which includes Medicaid and Maternal and Infant Care projects money), it is likewise estimated that in 1965 16.5% of the population lived in posttransfer poverty households, while in 1972 this incidence changed to 11.9% (Plotnick and Skidmore 1975, p. 174).

A similar persistence over time may be observed in the extent to which lack of prenatal care is found among expectant mothers: although national trend data is not available, in New York City it may be noted that 10.6% of mothers of liveborn infants had received late (third trimester) or no prenatal care in 1951. By 1955, this figure had increased to 15.6% (Baumgartner and Pakter 1958, p. 939). In 1968, the corresponding figure was 16.3% (Kessner, et al. 1973, p. 74).

Thus, indications for a lack of change in many of the variables discussed above may be noted, although testing of hypothesized changes in relationships between these variables is certainly a topic for future research. It would be englightening, for example, to observe the extent to which changes induced by health and welfare programs such as Medicaid, or the neonatal intensive care programs, may have worked to alter the relationships estimated in the present paper between poverty, race, and infant mortality. In this respect, it is hoped that estimates of relationships made from the present data will prove useful as baseline data for future studies.

APPENDIX A

Defining Income Poverty

Guidelines for defining income poverty in the present research are based upon those published by the Community Services Administration (CSA 1975). These original criteria were adjusted for changes in the standard of living between 1965-75, as reflected in changes in the Consumer Price Index. Adjustments were made according to instructions in the CSA report cited above (pp. 1-2).

Two changes in definition were made to better suit the research reported here: 1) Since farm-nonfarm family status was not an item asked in the followback surveys, all respondents were classified as either in or out of poverty upon the basis of nonfarm guidelines. 2) The income guidelines were adjusted downward so that approximately the lower fifth of the population studied was classified as being in poverty—perhaps better termed extreme poverty. For a family of three, this meant an approximate reduction in the income cutoff point of from $3,000 to $2,000 (1965 dollars).

Since the followback survey coded income units into categories (i.e., $3,000-$3,999), the midpoints of these intervals were used to derive the guidelines listed below:

Family Size	Family Income
1 or 2	less than $1,000
3 or 4	$1,000 - $1,999
5 or 6	$2,000 - $2,999
7 or 8	$3,000 - $3,999
9-12	$4,000 - $4,999
13 or more	$5,000 - $6,999

In both surveys, family income was defined as follows:

. . . family income refers to the total of all income received during the calendar year prior to the year during which the birth or the infant death occurred. Family income was defined to include all income received by the mother and by all persons related to the mother by blood, marriage, or adoption, and living in the same household at the time of birth. Income from all sources such as wages, salaries, unemployment compensation, rent, interest, dividends, help from relatives, profits and fees from own business or farm, welfare payments, social security payments, and insurance proceeds were asked for. [NCHS 1972, Appendix II].

APPENDIX B

Estimating the Impact of Factors

In regard to the procedure used to make rough estimates of the impact of factors affecting, for example, HOSP, the following is known:

1. 45 infants in the white sample were not born in a hospital.
2. Of all white infant births in the sample, 907 were born into poverty, 5,279 were not.
3. The rate of nonhospitalization for poverty infants is approximately 3.7 times that for nonpoverty infants.

Thus, we may write the following equation:

$$45 = 3.7x(907) + x(5279),$$

where x = the rate for nonpoverty infants. Solving, we obtain

$$x = .0052$$

$$3x = .0193,$$

implying the excess rate is .0141. Since our sampling fraction is 1/1,000, we have (907)(.0141)(1,000) ≈ 13,000 infants, which due to poverty, are not born in a hospital.

An exact procedure can be derived as follows: If $\hat{P} = \hat{\gamma}^B_j \ldots \hat{\gamma}^N_n$, and $\hat{\gamma}^A_1$ = the estimated effect of being in income poverty upon the expected death rate, and conversely $\hat{\gamma}^A_2$ = estimated effect of not being in income poverty, then the partial effect of income poverty upon the expected death rate is to multiply $\hat{P}$ by 1.2; this gives $\hat{DR}_1 = 1.2\hat{P}$. The partial effect of no income poverty is to multiply $\hat{P}$ by 1/1.2; this gives $\hat{DR}_2 = (1/1.2)\hat{P}$. If we take $\hat{DR}_1 - \hat{DR}_2$, we have the excess expected death rate for these particular categories of the variables j . . . n. If we multiply the estimated number of individuals who are in poverty and in

cell j . . . n of the cross-classification by this excess rate, we get the estimated number of deaths due to poverty for this one cell. Doing such calculations over all cells will give the total estimated number of deaths attributable to poverty. In notational form, this would be:

Expected number of deaths due to poverty =

$$\sum_{j \ldots n} (\hat{\gamma}_1^A - \hat{\gamma}_2^A)(\hat{\gamma}_j^B \ldots \hat{\gamma}_n^N)\hat{f}_{1j \ldots n}.$$

A major problem with this approach, however, is the accumulation of error as the different estimates are multiplied.

NOTES

[1]One disconcerting thought here is that certain death processes which are quite rare will never be suitably studied because of a lack of observational data.

[2]ECTA, a program to calculate log-linear fits for hierarchical models for contingency tables, is available from Professor Leo A. Goodman, Department of Statistics, University of Chicago.

[3]Our samples are of births and deaths, and thus our dependent variables are in fact expected central death rates for a particular cell of a cross-classification table. The numerators for these expected neonatal and postneonatal rates are thus the numbers of neonatal and postneonatal deaths. Our models assume that the exposed population is in each case the same--the total number of live births in that particular cell. Strictly speaking, the exposed population for the postneonatal period would be better approximated by the live births minus the neonatal deaths. With our data, however, this amounts to a change of only approximately 120 (out of 7,000) exposed infants. For simplicity, we will thus ignore this imprecision in the estimation procedure.

[4]These significance tests are not adjusted to take into account the large number of tests made; this constitutes yet another reason to be conservative in our acceptance of the significance of certain estimates.

REFERENCES

Anderson, Odin W. 1958. Infant mortality and social and cultural factors: historical trends and current patterns. In Patients, physicians and illness, ed. E. Gatly Jaco, pp. 10-24. New York: The Free Press.

Antonov, A.N. 1947. Children born during the siege of Leningrad in 1942. Journal of Pediatrics 30:250.

Apgar, Virginia, ed. 1970. Down's Syndrome (Monogolism). Annals of the New York Academy of Sciences 171:303-688.

Baumgartner, L., and Pakter, T. 1958. Challenge of fetal loss, prematurity and infant mortality--assessing the local situation. Journal of the American Medical Association 167:936-44.

Benjamin, B. 1965. Social and economic factors affecting mortality. Paris: Mouton and Co.

Bennett, Doris. 1976. Five years at Columbia Point Health Center: a high level of nonachievement. Radcliffe Quarterly 62(3):4-7.

Bergner, L., and Susser, M.W. 1970. Low birthweight and prenatal nutrition: an interpretive review. Pediatrics 46:946+.

Bishop, Yvonne M.M., and Mosteller, Frederick. 1969. Chapter IV-3, "Smoothed contingency table analysis," In The National Halothane Study, eds. John P. Bunker et al., National Institute of General Medical Sciences, National Research Council Subcommittee on the National Halothane Study, Bethesda, Maryland. Washington, D.C.: Government Printing Office.

Bishop, Yvonne M.M.; Fienberg, Stephen E.; and Holland, Paul W. 1975. Discrete multivariate analysis: theory and practice. Cambridge: MIT Press.

Blau, Peter, and Duncan, Otis D. 1967. The American occupational structure. New York: John Wiley and Sons.

Chase, Helen. 1964. The relationship of certain biologic and socio-economic factors to fetal, infant and early childhood mortality, part A. Washington, D.C.: The Children's Bureau.

Community Services Administration. 1975. CSA income poverty guidelines (revised). CSA Instruction No. 6004-1g. Washington, D.C.: Community Services Administration.

Duncan, Otis D. 1966. Path analysis: sociological examples. The American Journal of Sociology 72 (1):1-16.

_____. 1968. Inheritance of poverty or inheritance of race? In Understanding poverty: perspectives from the social sciences, ed. Daniel P. Moynihan, pp. 85-100. New York: Basic Books.

Goldberger, Arthur S. 1964. Econometric theory. New York: John Wiley and Sons.

Goodman, Leo A. 1972. A modified multiple regression approach to the analysis of dichotomous variables. American Sociological Review 37:28-46.

_____. 1973a. The analysis of multidimensional contingency tables when some variables are posterior to others: a modified path analysis approach. Biometrika 60:179-92.

_____. 1973b. Causal analysis of data from panel studies and other kinds of surveys. American Journal of Sociology 78:1135-91.

Gortmaker, Steven L. 1977. Stratification, health care, and infant mortality in the United States. Ph.D. dissertation, University of Wisconsin-Madison.

Gruenwald, P. 1974. Pathology of the deprived fetus and its supply line. In Size at birth, Ciba Foundation Symposium 27, pp. 3-19. New York: Associated Scientific Publishers.

Gruenwald, P., Funakawa, H.; Mitani, S.; Nichinina, T.; and Takenchi, S. 1967. Influence of environmental factors on foetal growth in man. Lancet I:1026-29.

Habicht, J.P.; Yarbrough, C.; Lechtig, A.; and Klein, R.E., 1974. Maternal nutrition, birth weight and infant mortality. In Size at birth, Ciba Foundation Symposium 27, pp. 353-370. New York: Associated Scientific Publishers.

Haggerty, R.J.; Roghman, K.J.; Pless, I.B. 1975. Child health and the community. New York: John Wiley and Sons.

Kessner, David M.; Singer, James; Kalk, Carolyn E.; and Schlesinger, Edward R. 1973. Infant death: an analysis by maternal risk and health care. In Contrasts in health status, Vol. I. Washington, D.C.: Institute of Medicine, National Academy of Sciences.

MacMahon, Brian; Pugh, Thomas F.; and Ipsen, Johannes. 1960. Epidemiologic methods. Boston: Little Brown and Company.

Marx, Karl. 1967. Capital: a critique of political economy. In The process of capitalist production, ed. Frederick Engels. Trans. Samuel Moore and Edward Aveling. Vol. 1. New York: International Publishers.

Mechanic, David. 1968. Medical sociology: a selective view. New York: Free Press.

Morris, J.N., and Heady, J.A. 1955. Social and biological factors in infant mortality. Lancet February 12:343-49.

National Center for Health Statistics. 1972. Infant mortality rates: socioeconomic factors. Series 22, No. 14. Washington, D.C.: Government Printing Office.

_____. 1973. Infant mortality rates: relationships with mothers reproductive history. Series 22, No. 15, Washington, D.C. Government Printing Office.

National Office of Vital Statistics. Yearly volumes 1920, 1954, 1975 Vital Statistics of the United States. Washington, D.C.: Government Printing Office.

Nerlove, Marc, and Press, James S. 1973. Univariate and multivariate log-linear and logistic models. Santa Monica: Rand Corporation

Norman, John C., ed. 1969. Medicine in the ghetto. New York: Meredith Corporation.

O'Sullivan , J.B. et al. 1965. Aspects of birth weight and its influencing variables. American Journal of Obstetric Gynecology 92:1023-29.

Plotnick, Robert D., and Skidmore, Felicity. 1975. Progress against poverty: a review of the 1964-1974 decade. New York: Academic Press.

Rush, D.; Davis, H.; and Susser, M. 1972. Antecedents of low birthweight in Harlem, New York City. International Journal of Epidemiology (4):375-87.

Scheffé, Henry. 1959. The analysis of variance. New York: John Wiley and Sons.

Shah, Fardia K., and Abbey, Helen. 1971. Effects of some factors on neonatal and postneonatal mortality. Millbank Memorial Fund Quarterly 49, (1), Part I:33-57.

Shapiro, Sam; Schlesinger, Edward R.; and Nesbitt, Robert E.L., Jr. 1968. Infant, perinatal, maternal, and childhood mortality in the United States. Cambridge: Harvard University Press.

Slesinger, Doris P., and Travis, Harry P. 1975. A study of infant mortality in Wisconsin, 1969, from linked birth and death records: an application of log-linear models. Working Paper No. 75-15. Madison: University of Wisconsin Center for Demography and Ecology.

Smith, C.A. 1947. The effect of wartime starvation in Holland upon pregnancy and its product. American Journal of Obstetric Gynecology 53:599+.

Statistical abstract of the United States. 1974. Washington, D.C.: Government Printing Office.

Stockwell, Edward G. 1962. Infant mortality and socio-economic status: a changing relationship. Milbank Memorial Fund Quarterly pp. 101-11.

Susser, M.W.; Marolla, F.A.; and Fleiss, J. 1972. Birthweight, fetal age and perinatal mortality. American Journal of Epidemiology 96: 197-204.

Theil, Henri. 1971. Principles of econometrics. New York: John Wiley and Sons.

Willie, Charles V. 1959. A research note on the changing association between infant mortality and socio-economic status. Social Forces 37:222-25.

Willie, Charles V., and Rothney, William B. 1962. Racial, ethnic, and income factors in the epidemiology of neonatal mortality. American Sociological Review 27 (4):522-26.